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Serial No.: 09/753,008  
Filed: January 2, 2001  
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Amendments to the Specification:

On page 1, please amend the first paragraph as follows:

This application claims priority of and is a continuation of U.S. Application No. 09/385,752, filed August 30, 1999, now U.S. Patent No. 6,228,591, which claims priority of and is a continuation of U.S. Application No. 08/651,999, filed May 23, 1996, now U.S. Patent No. 6,031,088, the entire contents of which are hereby incorporated by reference.

On page 6, please amend the paragraph at lines 7-8 as follows:

Figure 5A-5G represents the nucleotide sequence (SEQ ID NO:6) of the *PKD2* gene and the deduced amino acid sequence (SEQ ID NO:7) for PKD2.

Please amend the paragraph on page 12, line 18, through page 13, line 11, as follows. Note that the phrase "Identification of Mutations" is underlined in the original text.

Identification of Mutations. The mutation in family 1605 was detected initially in RT-PCR template using the cDNA-based primers F11 (5'-GGGCTACCATAAAGCTTTG-3') (SEQ ID NO:8) and R11 (5'-GTTCATGTTTCGATCAGTTCT-3') (SEQ ID NO:9) (205 bp product) and confirmed in genomic DNA using F11 with intronic primer IR11 (5'-GGGCTAGAAATACTCTTATCACC-3') (SEQ ID NO:10) (201 bp product). The mutations in families 97 and 1601 were initially detected in genomic DNA using intronic primers IF1C (5'-GCCTCAAGTGTTCCACTGAT-3') (SEQ ID NO:11) and IR1 (5'-AGGTTTTTCTGGGTAACCCTAG-3') (SEQ ID NO:12) (362 bp product). Amplifications

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were performed in standard conditions with hot start. Products were labeled by  $\alpha^{32}\text{P}$ -dCTP incorporation, diluted and denatured in formamide buffer prior to electrophoresis. SSCA was performed according to published protocols (Orita, M., et al. Genomics 5:874 (1989)). Sequencing of purified PCR products was performed with either an ABI 373a or 377 automated sequencing apparatus using cycle sequencing with dye terminator chemistries according to the manufacturer's protocol. The PCR primers were used as sequencing primers and all products were sequenced in both directions. The mutation in family 97 results in the loss of a Bsr I site. Genomic DNA amplified with IF1C and IR1 and digested with Bsr I yields products of 261 and 101 bp in the normal allele. The mutation in family 1605 results in the loss of a Taq I site. Genomic DNA amplified with F11 and IR11 and digested with Taq I yields products of 105 and 96 bp in the normal allele. The SSCA conditions used to demonstrate the mutation in the IF1C-IR1 genomic PCR product in family 1601 were 6% acrylamide (29:1), 1X TBE, on a 20 cm gel run at 14°C and 100 V for 6 hours.